Sport, Exercise, and the Common Cold

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ABSTRACT: Upper respiratory illness may cause more disability among athletes than all other diseases combined. This paper presents the essential epidemiology, risks of infection, and transmission features of upper respiratory illness. Those who provide health care for athletes must understand the subsequent implications of an upper respiratory illness on sport performance and should be familiar with participation and clinical management guidelines for athletes with an upper respiratory illness. The literature suggests that regular, rigorous exercise increases both the incidence and severity of upper respiratory illness, yet the immune system appears to have a distinct level at which moderate exercise promotes optimum health. Although research indicates that upper respiratory illness infections are surprisingly reluctant transmitters, upper respiratory illness transmission may escalate during winter sports seasons. The impact of upper respiratory illness on selected pulmonary, cardiac, and skeletal muscle functions may lead to illness complications in athletes, and sport performance during illness may also decline. Athletes should monitor symptoms, adjust training schedules, and rest during an upper respiratory illness.

Some researchers[53] contend that upper respiratory illness causes more acute disability among athletes than all other diseases combined. The average adult has between one and six episodes of a common cold each year,[5] but athletes who engage in heavy training and competition may suffer from more frequent colds.[42] Disease patterns among summer and winter Olympic athletes are remarkably consistent, with respiratory infections heading the list, followed by gastrointestinal disorders, and skin infections.[30] In the 1992 Winter Olympics, some of the world's greatest athletes were unable to compete or did not perform strongly because of an upper respiratory illness,[42] and several athletes were reportedly unable to compete in the 1988 Summer Olympic Games due to infectious illness.[23] It is important to understand, then, the essential epidemiology, risks of infection, and transmission features of upper respiratory illness. It behooves those who provide health care for athletes to realize the subsequent implications of upper respiratory illness on sport and exercise participation and performance. Health care providers must be familiar with participation and clinical management guidelines for athletes with an upper respiratory illness.

EPIDEMIOLOGY OF UPPER RESPIRATORY ILLNESS

There is support in the literature that regular, vigorous exercise (eg, sport participation) increases both the incidence and severity of upper respiratory illness. To date, 10 studies have made an attempt to examine the relationship between the epidemiology of upper respiratory illness and physical activity.[58] However, all 10 studies required subjects to self-report upper respiratory illness symptoms, and reported scores were subsequently used to diagnose whether or not an upper respiratory illness was present. This type of reporting needs to be interpreted cautiously because subjective rather than valid objective measurements are collected.

Better designed studies, done primarily with runners, have revealed an increased upper respiratory illness incidence rate as a result of physical activity. Heath et al[31] studied illness patterns longitudinally in a cohort of 530 male and female runners. An upper respiratory illness was indicated by a runny nose, cough, and/or sore throat. The results suggested that running mileage greater than 485 miles in a year was a significant risk factor for upper respiratory illness in this group. Peters and Bateman[48] studied the effect of the acute stress of running on infectious illness, showing that ultramarathon runners (35 miles) were at more than twice the risk of developing a upper respiratory illness within 2 weeks after competition. Additionally, symptoms were found to be most common in runners with the fastest race times.

Linde[38] investigated a 12-month upper respiratory illness incidence in a group of 44 elite runners and 44 nonathletes. Subjects were matched for age, gender, and occupational status. On average, the runners had 2.5 upper respiratory illnesses per year versus 1.7 upper respiratory illnesses in the control group. The average lengths of the illness periods were 7.9 and 6.4 days, respectively.

Results of a study conducted by Nieman et al[45] revealed that the risk of an infectious episode is five times greater for runners 1 week after a marathon race than for runners who trained but did not compete in the race. Another study completed by Nieman et al[44] investigated the incidence of upper respiratory illness in a group of recreational runners during January and February. At the time of the study, runners were training for either 5 km, 10 km, or half-marathon road races to be held in March. Results showed that 25% of those runners training more than 25 km/wk with an average of 42 km/wk reported at least one upper respiratory illness incident. On the other hand, 34.3% of the runners training less than 25 km/wk with an average of 12 km/wk did not report any incidence of upper respiratory illness. Nieman concluded that training more than 25 km/wk with the average mileage nearing 42 km/wk can increase the incidence for an upper respiratory illness.

RISKS OF UPPER RESPIRATORY INFECTION

In research reviews completed by Shephard et al[54] and Keast et al,[36] a plethora of literature is reported on the human and

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animal immune responses to exercise and stress. Included are countless studies that examine the relationship of exercise to antibody-mediated cell immunity, cell-mediated immunity, factors that modify immune mechanisms, phagocytic cells, numbers of circulating lymphocytes, lymphocytic function, catecholamines, glucocorticoids, and prostaglandins. Although very few definitive conclusions are drawn, this research indicated that the results of epidemiological studies on exercising individuals reported above can be supported. Nieman et al[45] stated that intense exertion, whether short-term and maximal or long-term and submaximal, may be associated with some potentially negative immune system changes; yet other research, also reported by Nieman et al,[44] suggested that moderate submaximal exercise bouts and long-term training may enhance immunosurveillance, potentially decreasing the risk for infection. Nieman[42] maintained that the relationship between exercise and upper respiratory illness can be depicted as a J curve with the most sedentary at greatest risk of upper respiratory illnesses along with the vigorously active; those engaged in moderate levels of activity manifest the apparently better host defense. Berk[6] suggested that the immune response is damaged by the stress of acute, exhaustive exercise. Lewicki et al[37] commented that nonspecific immunity is suppressed by intensive exercise and may render athletes more susceptible to infections. Fehr et al[21] and others[26,37,49] contended that immune suppression may also occur as a result of daily training over a long period of time (ie, overtraining). The defense mechanisms do not respond in the usual way (by elimination of the antigens); thus, a partial breakdown may occur periodically, leading to an acute infection.

Berk[6] surmised that people who exceed their optimum exercise level may be fostering infection. Though triathalons may not be too much for some, even moderate exercise may be detrimental for sedentary people. Hormones (eg, adrenaline, cortisol) and neuropeptides (eg, endorphins, encephalins) released from the stress of excessive physical activity, or from psychological or emotional stress, seem to have an adverse affect on the immune system.[1,12,20,25,36,47,57] Certainly, athletes who superimpose the psychological and emotional stress of competition upon a compromised immune system brought on by overexertion may be more susceptible to infection. Regardless of conditioning, those who exceed their physical limits are at risk to become sick, although the optimum level of exercise for the immune system is not known. More research is needed to improve our understanding of the workload threshold below or above which exercise becomes detrimental rather than protective. Tomasi[56] also contended that there appears to be a distinct level of moderate exercise for the immune system of each individual.

One other risk factor for an upper respiratory illness, the acute phase response, has been discussed by Heath et al.[32] The acute phase response following endurance exercise involves complement system, neutrophils, macrophages, various cytokines, and acute phase proteins. The acute phase response can last for several days, promoting clearance of damaged tissue and setting the stage for repair and growth. The authors suggested that the activity of the immune system in the muscle tissue repair and inflammation process means that resistance to respiratory infection is, perhaps, compromised. Further research is warranted.

COMMUNICABILITY AND TRANSMISSION OF UPPER RESPIRATORY ILLNESS

Specific research on the communicability and transmission of an upper respiratory illness has not been conducted on an athlete population. To date, research in this area has yielded contradictory results. The amount of virus being shed and the length of exposure time to the virus seem to be the center of this controversy. Although many families of viruses and their serotypes may cause the common cold, most upper respiratory illnesses are caused by rhinoviruses,[11] accounting for about 40% of all infections in adult populations.[9] There are more than 100 serotypes of the rhinovirus that may cause the common cold (see Table).[51] Rhinoviral infections occur throughout the year with well-defined periods of prevalence in the fall and spring, but the infections also can be found during the winter months.[9] Another group of viruses responsible for common colds among adults are the coronaviruses,[33] with the greatest incidence reported in persons between the ages of 15 and 19 years.[9] The greatest frequency of coronavirus infection is in the late fall, winter, and early spring, and these viruses are considered to be the major cause of winter colds.[9] Of particular significance to athletes in rigorous training are the enteroviruses, usually occurring during summer and autumn months. Although these viruses do not commonly cause acute adult respiratory tract illness,[41] the chief importance of enterovirus infection for the athlete lies in the association of some Enteric Cytopathogenic Human Orphan (ECHO) virus and Coxsackie virus strains with myocarditis and aseptic meningitis.[53] Exercise may increase the risk of developing enterovirus cardiomyopathy.[8,56]

The majority of viruses enter the body via the respiratory tract, then enter individual cells by penetrating the cell membrane and displacing host control mechanisms. The cell may produce many viruses that are then released by either cell lysis or by budding from the cell. This method of reproduction

Common Respiratory Viruses and Their Clinical Features

Virus Groups	No. of Serotypes	Clinical Features
Rhinovirus	>110	Common cold
Coxsackie A	23	Upper respiratory tract infection
Coxsackie B	6	Infection, especially common cold
Echovirus	31	Common cold
Adenovirus	33	Upper and lower respiratory tract infection
Coronavirus	7	Common cold
Respiratory syncytial virus	1	Upper respiratory tract infection: bronchiolitis, pneumonia
Influenza $(ABC)_3$	3	Influenza: upper respiratory tract infection
Parainfluenza	1	Upper respiratory tract infection
Herpesvirus: Epstein-Barr virus	1	Infectious mononucleosis

Reprinted from Roberts.[51]

renders the virus resistant to all the common antibiotics. The host responds to the viral infection by mobilizing antibody and cellular defenses. It is only if these defenses are overwhelmed that severe illness occurs.[51]

Although specific research on the communicability and transmission of upper respiratory illness has not been conducted on an athlete population, logical inferences from results of related work can be made about this group. Research indicates that rhinoviral infections are surprisingly reluctant transmitters and seem to be spread chiefly by aerosol contact, rather than by fomites or personal contact. It follows then that upper respiratory illness transmission among infected athletes to other team members would be spurious. Upper respiratory infections are spread from person to person by respiratory secretions containing a virus. The virus may gain entry to a susceptible host's respiratory tract via small or large particle aerosols, by direct contact, or by indirect contact involving contaminated environmental objects. Very low transmission rates (0% to 9%) have been reported for exposure periods extending from 45 to 72 hours, whether exposure was by aerosol alone or by all routes.[34] In a week-long experiment with childless married couples,[13] a transmission rate of 38% occurred between rhinovirus-infected donors and recipient spouses. Successful transmission was associated with donors who spent many hours with their spouses (122 hours), had virus on their hands and anterior nares, were at least moderately symptomatic, and had large amounts of virus in their nasal secretions.

Transmission rates of upper respiratory illness have also been investigated using a system called the Miniature Field Trial.[34] Natural rhinovirus transmission, theoretically by all possible routes, was achieved at predictable rates over time periods of up to 1 week. This system used experimentally induced adult donors, selected from a pool of infected individuals for their moderate to severe colds, and recipients. Interaction between donors and recipients took place in a single large room. In a series of miniature field trial experiments, the rate of transmission correlated closely with the number of hours the recipients interacted with the donors. About 200 hours of exposure to an individual with a moderately severe cold was needed for an antibody-free adult to have a 50% chance of infection. Other miniature field trial experiments examined aerosol and direct or indirect contact transmission of rhinovirus colds. Laboratory-infected men and susceptible men played cards together for 12 hours. In three experiments, the infection rate of restrained recipients (who could not touch their faces and who could only have been infected by aerosols) and that of unrestrained recipients (who could have been infected by aerosol, by direct contact, or by indirect fomite contact) was not significantly different. In a fourth experiment, transmission of fomites via playing cards heavily used for 12 hours by eight donors, represented the only possible route of spread. No transmissions occurred among the 12 recipients.[15]

Other research on upper respiratory illness transmission may imply that athletes could spread the virus through athletic equipment and implements. There is some inferential evidence that transmission by indirect contact routes is possible, but the importance of this evidence in the natural spread of colds is questionable.[27,34] The environment of an individual with a cold does become contaminated with rhinovirus. Virus readily gets onto the hands if the individual has a moderate to severe cold.[13] However, very little virus appears to be transferred from the hands. Virus was recovered from only 6 of 40 objects,[50] and 7 of 114 objects[29] recently handled by infected persons. All objects yielded very little virus. These researchers concluded that the spread of colds is unlikely to occur via objects contaminated by the hands of the infected person. Although transmission routes for an upper respiratory illness may not be completely clear, the potential for an infected individual to spread a cold appears important. Large amounts of virus are found to be shed by an infected individual for at least 8 days.[15] Virus may continue to be produced for 2 to 3 weeks.[18] Intensive studies with children in natural settings also indicate secondary attack rates (a week later or longer) of about 50%.[15] Subsequently, adults who live in households with children tend to suffer more colds per year, and adult women tend to suffer more colds than adult men.[28] For now, athletes should be advised to limit or avoid exposure to infected teammates or individuals. Studies designed to investigate specific transmission rates among different sporting activities (eg, wrestling and basketball) may assist in the development of intervention strategies.

SPORT/EXERCISE PARTICIPATION AND UPPER RESPIRATORY ILLNESS

Several investigators have examined the impact of upper respiratory illness on selected pulmonary, cardiac, and skeletal muscle functions. Because previous studies have rarely determined etiology of upper respiratory illnesses, caution should be taken when interpreting their results. The implications for continued sport and exercise participation relative to illness complications and susceptibility need to be considered. Protracted courses of upper respiratory illness and performance levels during illness also warrant discussion.

Three studies of the effects of upper respiratory illness on the pulmonary function of subjects at rest were completed in the 1970s, but investigation of the effects of upper respiratory illness on pulmonary function during exercise is needed. All three studies suggested that peripheral airway abnormalities are associated with upper respiratory illness. One study concluded that large airways were involved during upper respiratory illness.[46] This research demonstrated significant impairment of peak expiratory flow rate, forced vital capacity, forced expiratory volume in 1 second, and maximal midexpiratory flow rate, measured at 50% of vital capacity. Changes in the maximal expiratory flow rate measured at 75% of vital capacity were not significant. The other two investigations identified no large airway dysfunction. One of these found that the subjects developed increased frequency dependence of compliance,[7] and the other found a reduction in steady-state carbon monoxide diffusing capacity.[10]

Respiratory muscle strength was studied in 12 subjects who developed naturally acquired upper respiratory illness.[40] Maximum static respiratory and expiratory mouth pressures fell significantly during these infections. The greatest falls were

documented between the third and seventh days of clinical illness. However, the lowest pressures occurred several days after the peak of clinical symptoms, when malaise had greatly improved. Full recovery occurred by day 14. The authors concluded that weakness of the inspiratory muscles may contribute to breathlessness during exertion. In contrast, weakness of the expiratory muscles might affect the cough mechanism and clearing of pulmonary secretions. The authors speculated that those who suffer either from lower respiratory tract infections or from exercise-induced asthma should also refrain from athletic activity during upper respiratory illness or episodes of exercise-induced asthma.

Reduced functional capacity of skeletal and cardiac muscle has been demonstrated during upper respiratory illness. In a controlled test, Astrom et al[3] examined muscle tissue obtained from patients recovering from recent viral or mycoplasma illnesses. They found significantly reduced muscle enzyme activity (glyceraldehyde phosphate, lactate dehydrogenase, cytochrome oxidase, and citrate synthetase) in infected patients. Moreover, electron microscopy showed abnormalities in muscle ultrastructure. These changes had almost completely resolved when muscle biopsy was repeated 3 months after illness. Roberts[51] suggested that a decrease in muscle glycogen use occurred during upper respiratory illness, while Ardawi[2] reported that a decrease in muscle glutamine release occurred with upper respiratory illness during prolonged physical training. Other researchers[60] have also reported that myositis ossificans may be the result of hematoma infection following a respiratory tract infection.

The effects of myalgia and fever on muscle and circulatory function have also been examined.[24] During, but not after, a fever, subjects exhibited decreased isometric and dynamic strength and endurance. Impairment could not be explained by altered activities of relevant muscle enzymes or altered muscle ultrastructure. However, severity of myalgia, as rated by each subject, correlated significantly with reduced muscle function. Cardiac stroke volume was lower during and after a fever. During a fever, an increased heart rate maintained cardiac output at preinfection values, whereas cardiac output fell in early recovery. This decrease in cardiac output correlated significantly with the severity of the fever. The actual influence of a fever and myalgia from an upper respiratory illness on the above parameters has not been determined.

A variety of illness complications may be associated with upper respiratory illness, including protracted courses of infection and sudden death. Roberts[51] discussed an increasingly recognized postviral fatigue syndrome (epidemic myalgic encephalomyelitis). It usually occurs after a Coxsackie virus infection,[4] although it has also been diagnosed after influenza and varicella virus infections. The patient complains of persistent malaise, fatigue, lassitude, and aching muscles. Symptoms may last for months or years, and there is no treatment.

The predilection of the Coxsackie virus to produce myocarditis or pericarditis may increase the risk of acute arrhythmias leading to sudden death.[51,55] In a study of 78 sudden deaths during or immediately after exercise, Jokl and McClellan[35] found a history of recent upper respiratory tract infection in five subjects; cardiovascular problems accounted for most of the remainder. Roberts[51] commented that there are numerous anecdotal reports of death in young healthy people who undertake vigorous exercise during viral illness. He also reported that numerous case studies have identified viral infections as the cause of sudden death.

The impact of upper respiratory illness on sport performance has not been clearly identified. In related work by Friman et al,[24] a decrease in muscle performance correlated to the subjects' own ratings of the intensity of some disease-related symptoms such as myalgia, but not to a fever reaction. Regarding performance, then, the authors concluded that a person's perception or experience of a febrile illness seems to influence his ability and/or willingness to perform exercise. Roberts[52] presented four case reports of athletes who experienced a loss of form (decreased stamina, inability to manage normal training schedule) during subclinical episodes of upper respiratory illness. Two of these highly trained athletes had no prodromal symptoms, and two had minor symptoms of the upper respiratory tract. All had laboratory evidence of recent viral infections. Roberts[52] concluded that inquiry about recent minor illness should be standard practice in athletes with unexplained loss of form. Infections that are subclinical in the normal population may greatly affect maximum performance in athletes.

One study[59] has been completed concerning the reporting behaviors and activity levels of intercollegiate athletes with upper respiratory illness. This study attempted to discern which upper respiratory illness symptoms are the most problematic for an athlete. Distinctions among symptoms were assessed by examining which symptoms athletes reported the earliest to their medical supervisors (eg, athletic trainers, team physician) or coaches. Likewise, those cold symptoms, which prevented an athlete from participating in a practice or a game and/or affected perceptions of physical performance, were also examined. This study is the first to use a comprehensive and validated symptom checklist in sports medicine research. In addition, the study was further strengthened by specifying that an upper respiratory illness was only present in any athlete with three or more symptoms from this checklist. Similar studies employing the use of a self-report cold symptom survey have used the presence of only one symptom of a list of three as the determination of a cold. Symptoms of cough, fever, laryngitis, aching joints/muscles, and nasal discharge were significantly correlated with reporting behaviors, activity levels, and/or perceived physical performance ($p < .05$).

The impact of upper respiratory illness on sport performance has not been clearly identified. Certainly alterations in cardiac, respiratory, and skeletal muscle functions discussed above may individually or collectively alter performance, but further research is needed.

PARTICIPATION AND CLINICAL MANAGEMENT GUIDELINES

There has been no research regarding the disposition of an athlete with an upper respiratory illness. If the athlete has symptoms of a common cold with no constitutional upset, Roberts[51] recommended safely resuming training a few days

after the resolution of symptoms. However, if the athlete experiences symptoms or signs of extreme tiredness, myalgia, or swollen lymph glands, then he/she should not resume full training for at least a month. For very competitive athletes who cannot afford to miss any training days, even when ill, Eichner[19] recommended that athletes perform a "neck check." If symptoms are located "above the neck," such as a stuffy or runny nose, sneezing, or scratchy throat with no constitutional symptoms, then the athlete should be allowed to proceed cautiously through his/her scheduled workout at half the speed. After a few minutes, if the congestion clears and the athlete feels better, then intensity can be gradually increased. If the athlete feels worse, rest is recommended. The athlete with "below the neck" symptoms, such as a fever, aching muscles, hacking or a productive cough, vomiting, or diarrhea, should not train. Fitzgerald[22] comments that exercising during the incubation period of an infection may worsen the illness. Certainly, athletes who feel that they may be getting ill should reduce their training schedule for 1 or 2 days. If exercise is capable of compromising the immune response in healthy subjects, it seems logical to assume that exercise would certainly do so during an illness. If this is the case, both symptom severity and duration may be increased. In addition, training techniques should take into consideration the need for the body to restore host resistance by including lower intensity training interposed between higher intensity training bouts.[32] Again, further research in this area is needed.

According to comments found in selected medical magazines and newsletters, heart rate and oxygen consumption changes that accompany a fever during some upper respiratory illnesses may provide reason to decrease training. Heart rates increase by 2.44 beats/min with every 1.5°C rise in temperature in afebrile subjects.[39] As discussed earlier, cardiac output correlated significantly with the severity of the fever.[24] A fever also increases the demand for oxygen.[16] For every increase of 1°C over 37°C, there is a 13% increase in oxygen consumption. In addition, overtraining, fatigue, or illness may increase resting heart rates. For instance, a difference of 10 to 20 beats per minute upon rising in the morning may signal the onset of illness or lack of adequate rest between workouts.[48]

Nieman[42] advocated several precautions that can help athletes reduce their risk of an upper respiratory illness. The athlete is urged to eat a well-balanced diet, keep other life stresses to a minimum, avoid overtraining and chronic fatigue, shun sick people before and after important events, obtain adequate sleep, and space vigorous workouts and competitive events as far apart as possible. If the athlete is competing during the winter months, a flu shot is highly recommended.

Conditions that may increase the transmission of an upper respiratory illness among athletes warrant the attention of those in the sports medicine field. Particularly during winter weather, athletes are exposed to the cold virus in crowded dormitories, classrooms, and gymnasiums, perhaps accounting for the higher incidence of colds during cooler months.[9] Because there is some evidence that strenuous exercise may increase the incidence of upper respiratory illness infections,[17,32,38,44,49] athletes should be advised to maintain a good health profile (eg, rest, nutrition, stress management), especially during winter months. In clearing the nasal passages, facial tissues should be used and care should be taken by team members to clear their respiratory passages gently in order to prevent wide dissemination of infected mucous.[14] Casey et al[9] recommended careful hand washing, avoidance of direct skin-to-skin contact, or contact with contaminated tissues, sporting equipment, and appliances. Towels and water bottles should not be shared. Paper handkerchiefs and cups should be carefully used and disposed of in closed plastic container bags. Commonly used washing facilities should be cleaned with disinfectants or tincture of iodine before and after each use.

Treatment for viral upper respiratory illnesses is supportive for the most part, consisting of rest, fluids, analgesics, and over-the-counter cold remedies. Acetaminophen is recommended for fever, headache, and muscle pain along with lozenges, saltwater gargles, or viscous lidocaine for sore throat. If training is to be attempted, caution must be used with cold medications containing antihistamines because their anticholinergic side effects may lead to impaired thermoregulation. Also, some decongestants still contain substances banned by several governing athletic bodies (eg, ephedrine). Athletes must be careful to avoid potential disqualification by testing positive for an illegal substance from a seemingly innocent over-the-counter medication.

CONCLUSION

The essential epidemiological and immunological features of upper respiratory illness seem to indicate that sport and exercise participation may increase the incidence of upper respiratory illness, depending on the individual's immune system reaction. The risk of upper respiratory illness transmission among a cohort of athletes is potentially high, although the risk is not completely clear. Athletes' performance levels may decline during an upper respiratory illness; however, more research is needed in this area. Through early intervention and education programs, illness complications and protracted courses of upper respiratory illness may be prevented.

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